

## Cribra Orbitalia in Two Temporally Disjunct Population Samples From the Dakhleh Oasis, Egypt

SCOTT I. FAIRGRIEVE<sup>1\*</sup> AND J.E. MOLTO<sup>2</sup>

<sup>1</sup>*Anthropology Program and Department of Biology, Laurentian University, Sudbury, Ontario, Canada P3E 2C6*

<sup>2</sup>*Department of Anthropology, Lakehead University, Thunder Bay, Ontario, Canada P7B 5E1*

**KEY WORDS** cribra orbitalia; porotic hyperostosis; anemia; palaeonutrition; parasitism; third-intermediate; Romano-Christian; Egypt; Dakhleh Oasis

**ABSTRACT** Cribra orbitalia (CO), an osseous sign of anemic stress, occurs in 67% (n = 296) of the pre-Roman (n = 153) and Roman (n = 143) period crania from the Dakhleh Oasis, Egypt. CO is primarily a childhood condition in these samples, and its prevalence is significantly higher in virtually all cohorts in the pre-Roman group, including among females, who display higher rates of active lesions. This temporal trend suggests that the underlying causative factors (i.e., synergism between disease and nutrition) were less pervasive in the Roman period. In both population samples, anemic stress develops in some perinates prior to the expected minimum age for the development of iron deficiency anemia. This suggests additional causes of anemic stress in the Dakhleh population. A strong candidate is folic acid deficiency and its concomitant, megaloblastic anemia, which results from weaning of infants on goat's milk, a known practice in ancient Egypt. The putative incorporation of other food items in the weanling diet, particularly honey, a confirmed source of *C. botulinum*, represents yet another retrospective data source to help understand the epidemiological profile of cribra orbitalia in this population. Comparative data from other Egyptian populations, though limited, show similar patterns, however, they display a lower prevalence than the data from Dakhleh. *Am J Phys Anthropol* 111:319–331, 2000. © 2000 Wiley-Liss, Inc.

Cribra orbitalia (CO) is a bone lesion of the orbital roof purported to be an indicator of an anemic stress (e.g., Mensforth et al., 1978; Ortner and Putschar, 1985; Mittler and Van Gerven, 1994). This condition has traditionally been linked to an ectocranial vault lesion known as porotic hyperostosis. The etiological association of these two bone pathologies has been confirmed by Stuart-Macadam (1989). Currently, references in the literature to the orbital lesions are found in discussions of porotic hyperostosis. The support for this classification has a physiological basis; an anemic stress suffered in early childhood results in the expansion of

hemopoietic bone marrow to increase erythrocyte (red blood cell) production (Moseley, 1974; Stuart-Macadam, 1985, 1987).

Recording the prevalence of cribra orbitalia in osteological analyses is commonplace (e.g., Cohen and Armelagos, 1984). Studies

---

Grant sponsor: Natural Sciences and Engineering Council of Canada; University of Toronto Open Doctoral Fellowship; Laurentian University Research Fund; Social Sciences and Humanities Research Council; Grant number: 410-94-0857; Grant sponsor: Lakehead University Research Grants.

\*Correspondence to: Dr. Scott I. Fairgrieve, Forensic Osteology Laboratory, Subdepartment of Anthropology, Laurentian University, Sudbury, Ontario, Canada P3E 2C6.  
E-mail: sfairgri@nickel.laurentian.ca

Received 2 September 1997; accepted 10 October 1999.

tend to go beyond mere description by providing an epidemiological profile (age and sex distribution) of the condition. Generally, these lesions show greater severity and prevalence in subadult remains (e.g., El-Najjar et al., 1976; El-Najjar, 1976). Gender differences are not a consistent finding, although interpopulation variation is considerable. Various explanations have been invoked to the latter. For example, Angel (1964, 1971), in his classic studies of porotic hyperostosis in populations from Cyprus and Greece, suggested that these lesions reflect thalassemia genotypes. Using the Hardy-Weinberg model he attributed the variability to the allelic distributions of a hypothetical mating population. Underlying this model is the concept that heterozygotes for the thalassemia allele will survive longer and will be less severely affected than homozygotes who died before reproductive maturity. However, Angel (1971) cites the prevalence of porotic hyperostosis as approximately 20% regardless of severity, which is significantly less than the prevalence predicted from the Hardy-Weinberg equilibrium. El-Najjar et al. (1976), however, pose a dietary hypothesis, relating the high prevalence of CO in their American southwest samples to a dependence on maize. Walker (1986) found that porotic hyperostosis in his southern California Island populations varied according to the availability of fresh water supplies, with more stagnant conditions being highly correlated with increased prevalence of cribra orbitalia. He suggests that the condition is most likely of an infectious origin. Clearly, a multifactorial etiology would seem the most appropriate model for palaeoepidemiological studies of porotic hyperostosis, although in each environmental/population niche certain specific synergistic factors will predominate. Addressing potential etiological factors in specific niches is an elusive goal of current research.

The often quoted prevalence of these lesions in the literature, and its importance to palaeoepidemiological research, warrants the reporting of any situation where the proportion of affected individuals deviates greatly from other published values. This situation occurs in samples from the Dakhleh Oasis of Egypt's Western Desert. In

the population samples from two cemeteries in this Oasis, a combined prevalence of over 60% of the children show CO to varying degrees. Also, there is a temporal decrease in the prevalence of CO, which suggests that the etiological factors were changing. This paper provides a detailed account of CO and possible etiological factors in the Dakhleh Oasis populations.

## MATERIALS AND METHODS

### Skeletal sample

The Dakhleh Oasis (Fig. 1) is one of five major depressions in Egypt's Western Desert, measuring 100 km long from east to west, and 25 km at its widest point from north to south (Cook et al., 1988). It is located approximately 800 km south-southwest of Cairo, with a land area of 2,000–3,000 km<sup>2</sup> (Mills, 1979). The flat clay plains of the Oasis are made up of soil with a high iron oxide content (Churcher, 1983). Presently, the climate is extremely arid with an annual rainfall of 0.7 mm (Mills, 1979). The humidity rarely exceeds 50%, and the temperature ranges from a daily maximum average in January of 21.5°C to a July average of 39°C (Mills, 1979). The current population of the Oasis (approximately 35,000) is agriculturally based, and grows vegetables, fruits (dates, apricots, oranges, and olives) and cereals (rice, sorghum, and wheat). The vegetables are grown for local consumption and are supplemented from the Nile Valley in the hot weather (Mills, 1979). Agriculture, and indeed human existence, is made possible by the exceptional nature of the Nubian Sandstone and shale series, which contains one of the largest ground water reserves in the world. Today, as in the past, some water seeps to the surface, although the main access for human use is through artesian wells. The history of settlement patterns strongly suggest that the conditions in the Oasis were as arid in the past as they are today (Schwarcz et al., 1999). There are no contemporary records of past climates; however, it is generally known that the eastern Sahara was about as arid today as it was throughout late Pharaonic and Roman times (Butzer, 1976). Establishing the amount of surface water and rainfall in the past is a valuable research pursuit as it

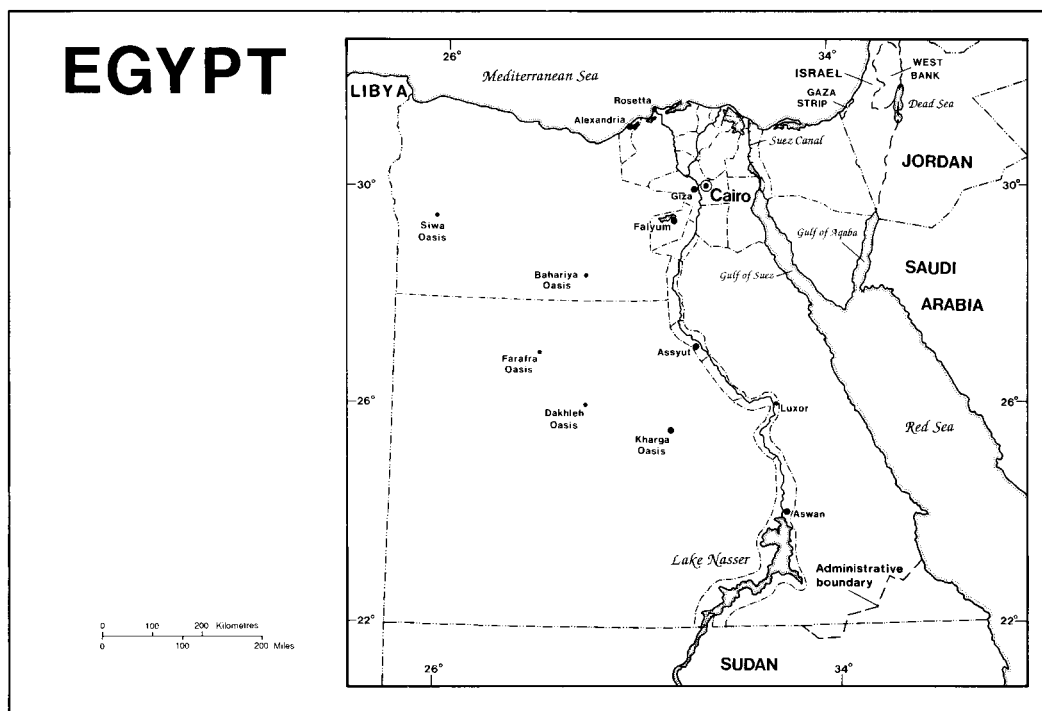


Fig. 1. A map of Egypt showing the position of the Dakhleh Oasis in the Western Desert.

has the potential to alter understanding of the epidemiological patterns of a population. It is known, however, that past agricultural subsistence pursuits were dominant throughout the Pharaonic Periods and coalesced during the Roman Period (Mills, 1984).

Since 1977, the Oasis has been the focus of the Dakhleh Oasis Project (DOP). The bioarchaeological component of the DOP has focused on the analysis of human remains from two cemeteries; 'Ein Tirghi (ET) and Kellis 2 (K2). 'Ein Tirghi (31/435-D5-2) has yielded remains of over 700 individuals from 58 tombs that date to the Late Period. The cemetery was used over a period of several hundred years from ca 900 BC to possibly early Ptolemaic times. Skeletons from only one tomb, E31, have been radiocarbon dated. These skeletons, 25 and 36, yielded respective calibrated dates of  $795 \pm 70$  BC and  $800 \pm 60$  BC (Isotrace TO-4476), which are both in the Late Third Intermediate Period (Molto, 2000). Hope posits that the majority of the ET samples would be in the Late

Period (C.A. Hope, personal communication, January, 1997), a view that requires further testing. This study involves crania from eight ET tombs, E31, E33, E34, E36, E37, E40, E45, and E52. The ancient town of Kellis, has two major cemeteries associated with it. The western cemetery, Kellis 1 (31/420-C5-1), predates the Christian Period (c. 60 BC–100 AD), while Kellis 2 (31/420C5-2), the focus of this study, is clearly within the Christian Period (c. 100 AD–400 AD) as illustrated by the classic Christian mortuary position (i.e., single inhumations with the body oriented east-west with the head in the latter position). Five calibrated radiocarbon dates on human skeletons from K2 (burials 5, 6, 94, 95, and 116) support the Christian Period affiliation of this sample. The sample of 278 burials excavated so far, date between 300 and 450 AD or during the final phase before the town of Kellis was abandoned. Therefore, the chronological separation of the cemeteries in this study potentially span some 1300 years. Geo-

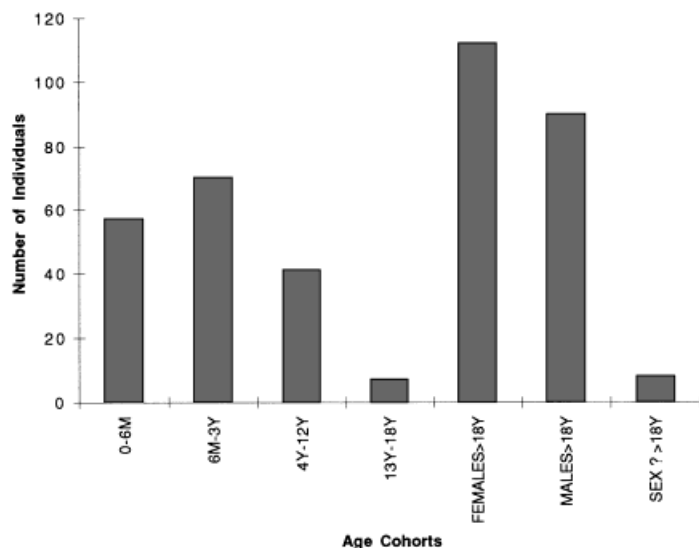


Fig. 2. Demographic profile of the Ein Tirghi and Kellis 2 cemeteries.

graphically these cemeteries are only approximately 15 km from one another.

The preservational quality of the bone in all samples is excellent. Extraction of Type I collagen from bone tissue and tooth dentin reflects the preservational quality through a high percentage yield (ca. 15–20%) by weight. This conclusion is also supported by an amino acid residue analysis (Fairgrieve, 1993). Additionally, the remains were complete in most cases, affording the opportunity to analyze the skeletons completely for vital statistics and pathology. These facts are important in order to present an accurate differential diagnosis of any condition that may be present.

The combined demographic profile of the Ein Tirghi and Kellis samples is presented in Figure 2. A composite sample of 385 skeletons were included in this study. Of that total, eight could not be ascribed either to an age or sex category due to damage suffered largely during episodes of tomb-robbing. A total of 377 were preserved well enough to be aged and sexed using conventional osteological methodologies (Bass, 1987). Specifically, age at death assessments were based on dental development (Ubelaker, 1978), and pubic symphysis metamorphosis (Katz and Suchey, 1986; and Brooks and Suchey, 1990). Sex determination of adult remains was based on cranial morphology

(Bass, 1987) and pubic symphysis morphology (Phenice, 1969). One member of the Dakhleh Oasis Project (M. Marlow) has developed a refined age sequence for the infants based on dental calcification. These data were used in conjunction with long bone length to age neonates. All of the aging methods utilized have their own standard deviations. An appreciation of this fact is particularly important due to the significance of infants to this study. Although Ubelaker's dental development standards for infants have a standard deviation of 3 months, it should be recognized that with a mean age of 6 months remains could be 3, 6, or 9 months of age. The importance of such an early onset of an anemic stress is not necessarily diminished by these factors. When combined with other aging methods our analysis of lesion distribution is still presented using age ranges.

#### Assessment of cribra orbitalia

Although a general assessment of all pathology present on the remains was conducted (Molto, 1986), a more detailed study of the prevalence and severity of CO was conducted by SIF. It is well established that there are as many methods for visually assessing these lesions as there are investigators (e.g., Nathan and Haas, 1966; Steinbock, 1976; Mensforth et al., 1978; Guidotti,

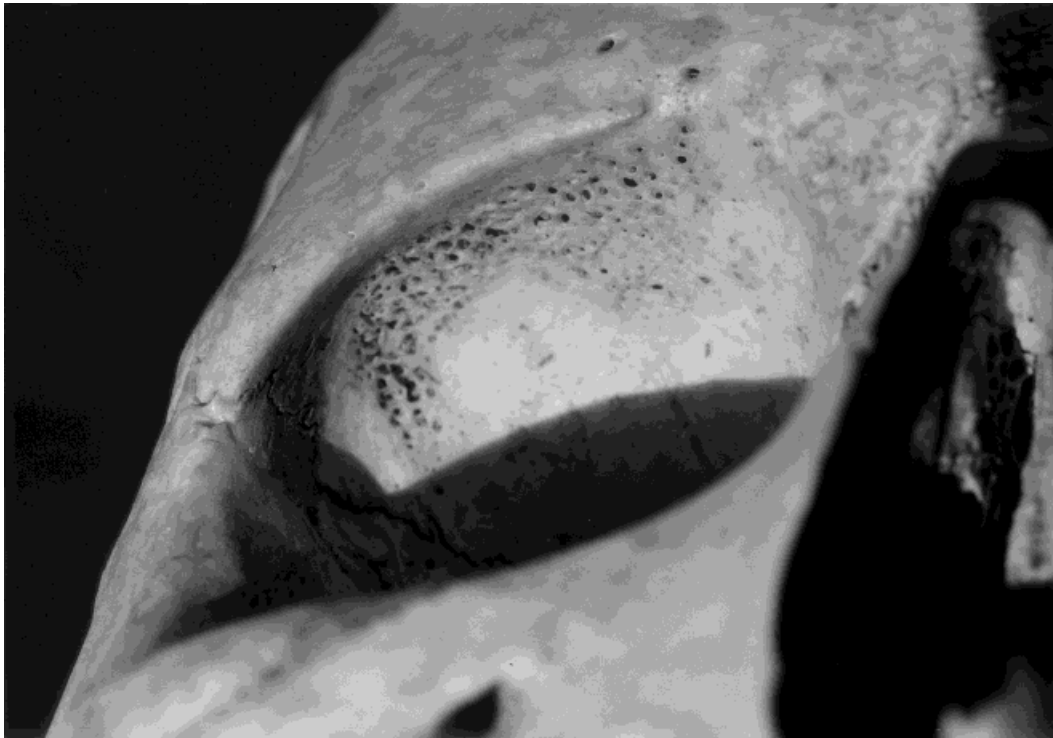


Fig. 3. Typical "healed" or inactive CO found in an adult from the Dakhleh Oasis, Egypt.

1984; Stuart-Macadam, 1982, 1985). However, for comparative purposes and expediency, the method outlined by Steinbock (1976), adopted from Nathan and Haas (1966) is used herein. The categories are defined as follows (Steinbock, 1976:239): the *porotic type* is "characterized by scattered fine openings affecting the roof of the orbit"; the *cribrotic type* shows "openings larger and more numerous, tending to coalesce into larger apertures"; and finally, the *trabecular type*, exhibits small openings coalescing into "large, irregular apertures often arranged in radiating patterns from one or more centers in the orbital roof." In addition, the lesions were recorded for their status at time of death [i.e., healed (Fig. 3) and reactive (Fig. 4)] following Mensforth et al. (1978). This recording system is consistent with the method reported by Mittler and Van Gerven (1994) in their study of the Medieval Kulubnarti remains. Both orbits were assessed in each case.

In order to interpret the epidemiological profile of this condition, statistical compari-

sons ( $\chi^2$ ) on the basis of age, sex and the assessed state of lesions were conducted.

## RESULTS

The combined data of cribra orbitalia relative to age, sex, and lesion status are summarized for both cemeteries in Table 1. Figure 5 depicts the percentage of active and healed lesions for each cohort of affected individuals including females and males over 18 years of age. The trend clearly indicates that active lesions are more prevalent in younger individuals, declining gradually in the older cohorts. This pattern characterizes both ET and K2 (Figs. 6 and 7, respectively). These data support the argument that cribra orbitalia is indicative of a childhood anemia (Stuart-Macadam, 1985). However, the overall prevalence of 66.9% (198/296) obscures a significant diachronic decrease.

Tables 2 and 3 summarize the prevalence of cribra orbitalia according to age, sex, and lesion status for the 'Ein Tirghi and Kellis 2 cemeteries, respectively.



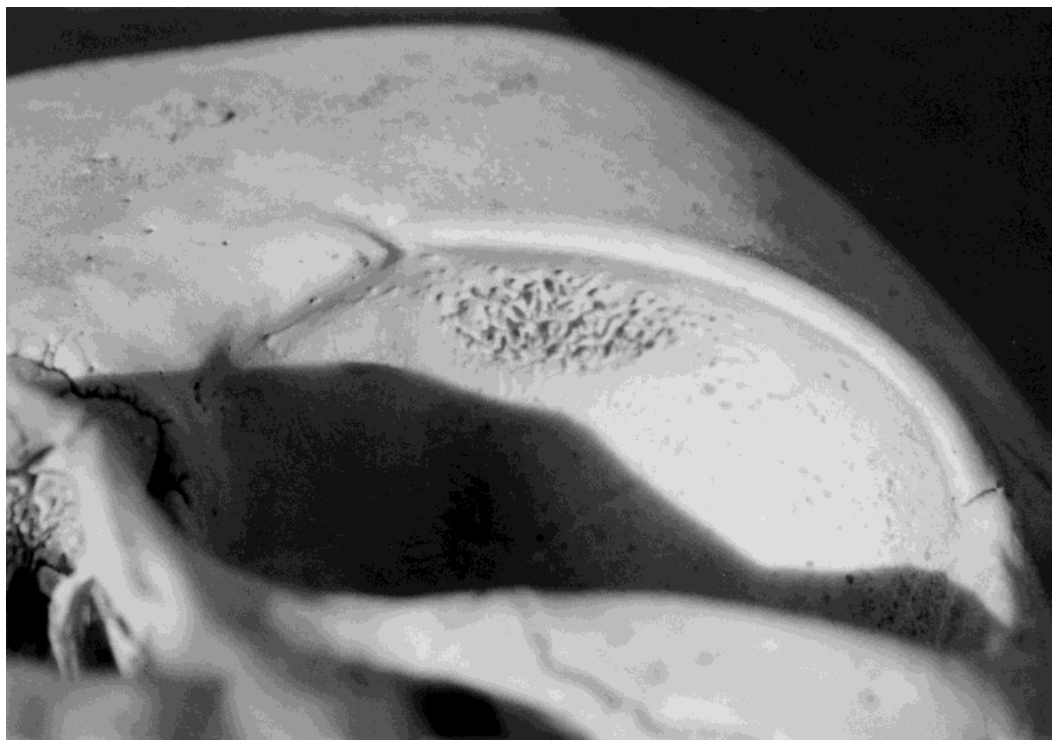


Fig. 4. Typical "active" CO found in a subadult from the Dakhleh Oasis, Egypt.

TABLE 1. Prevalence of cribra orbitalia and active lesions by age and sex from the 'Ein Tirghi and Kellis 2 cemeteries combined

| Age cohort                      | Total<br>N | Assess-<br>able | Cribra<br>orbitalia |        | Active<br>lesions |                     |
|---------------------------------|------------|-----------------|---------------------|--------|-------------------|---------------------|
|                                 |            |                 | n <sup>1</sup>      | %      | n <sup>2</sup>    | % of n <sup>1</sup> |
| 0-6 months                      | 57         | 36              | 10                  | 27.77  | 9                 | 90.00               |
| 6 months-3<br>years             | 70         | 48              | 39                  | 81.25  | 27                | 69.23               |
| 4 years-12<br>years             | 41         | 33              | 31                  | 93.94  | 18                | 58.06               |
| 13 years-18<br>years            | 7          | 5               | 5                   | 100.00 | 1                 | 20.00               |
| Females >18<br>years            | 112        | 96              | 58                  | 60.42  | 9                 | 15.52               |
| Males >18<br>years              | 90         | 74              | 52                  | 70.27  | 5                 | 9.62                |
| Sex ? >18<br>years <sup>3</sup> | 8          | 4               | 3                   | 75.00  | 0                 | 0.00                |
| Total                           | 385        | 296             | 198                 | 66.89  | 69                | 34.85               |

<sup>1</sup> Refers to the number of individuals with CO.

<sup>2</sup> Refers to the number of individuals with "active" lesions of CO.

<sup>3</sup> Skeletons of indeterminant sex over the age of 18 years.

The prevalence at 'Ein Tirghi is 78.4% (120/152) compared to 54.6% (78/152) at Kellis 2; a statistically significant difference ( $\chi^2 = 19.88$ , degree of freedom (df) = 1,

$P = 0.001$ ). It follows that whatever causative factors were in operation they were more pervasive in the earlier population. This assertion, however, requires a detailed specific investigation of these data, particularly in the younger age cohorts.

Individuals were included in the perinatal category if the remains were developmentally consistent with birth to 6 months in age. It is germane to note a discrepancy in sample size; 3 for ET and 33 for K2. All the neonates were affected in the former site compared with 21.2% (7/33) at K2. Though this result is statistically significant ( $\chi^2 = 5.035$ , df = 1,  $P = 0.0248$  with Yates' correction for continuity) the sample size disparity suggests a degree of caution.

The second cohort (6 months-3 years) duplicates the sample size differential (n = 31 for K2 versus n = 17 for ET) and the higher and statistically significant prevalence pattern (100% for ET and 70.6% for K2;  $\chi^2 = 4.318$ , df = 1,  $P = 0.0377$ ). Clearly, the probability of experiencing anemia in

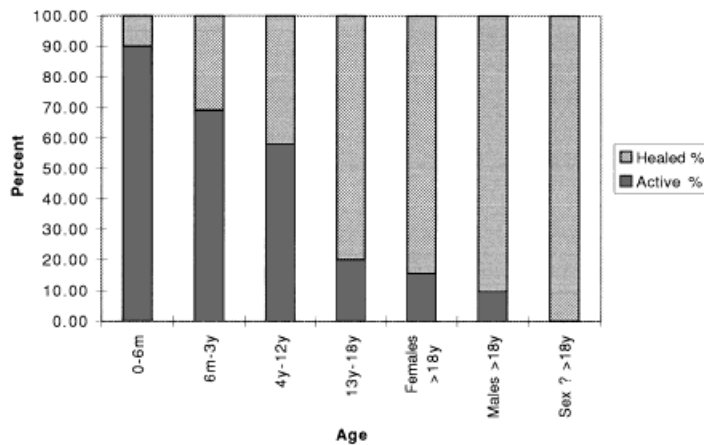


Fig. 5. Prevalence of active and healed lesions by age and sex for the 'Ein Tirghi and Kellis 2 Cemeteries combined.

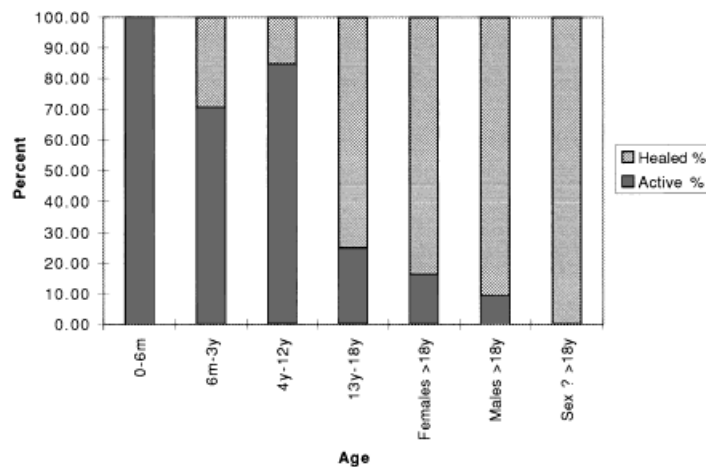


Fig. 6. Prevalence of active and healed lesions by age and sex for the 'Ein Tirghi Cemetery.

these early cohorts exceeds the probability of having normal hemoglobin levels.

In the final childhood cohort (4–12 years) no significant difference between ET and K2 occurs (100.0% and 90.0%, respectively). As is normal of paleodemographic profiles, there are fewer individuals in the next cohort (13–18 year olds), although the number is lower than expected (see Henneberg and Steyn, 1993). In total, only seven individuals recovered, while only five (four from ET and one from K2) could be analyzed for CO. While all five demonstrate CO, it is only active in one (from ET). The prevalence of CO in adult females (>18 years) is the same for ET and K2 (60.7% and 60.0%, respectively). In adult males (>18 years), those from ET have a significantly higher prevalence than those from K2 (84.3% (43/51)

versus 39.1% (9/23);  $\chi^2 = 13.403$ ,  $df = 1$ ,  $p = 0.0003$  with continuity correction). Of note is the fact that the prevalence of active lesions is higher in females in both population samples, although the males had a higher overall prevalence in ET and lower in K2.

Epidemiological trends for cribra orbitalia were also tested within each cemetery sample. In the case of ET, the prevalence of CO and age of the individual demonstrated a significant relationship ( $p = 0.0031$ ). Related to this trend is a significant relationship between the age and level of lesion severity ( $p = 0.0001$ ). However, there was no significant relationship with CO severity and prevalence with the sex of the individual. For K2, prevalence and age ( $p = 0.002$ ), and age and severity ( $p = 0.0001$ ) were statisti-

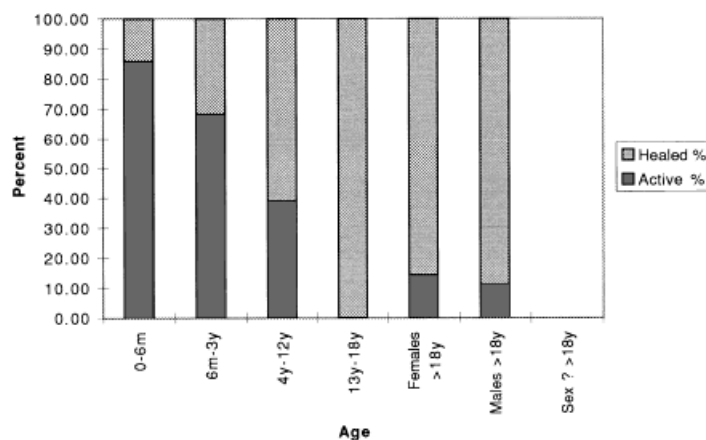


Fig. 7. Prevalence of active and healed lesions by age and sex for the Kellis 2 Cemetery.

TABLE 2. Prevalence of cribra orbitalia and active lesions by age and sex from the 'Ein Tirghi cemetery

| Age cohort                   | Total N | Assess-able | Cribra orbitalia |        | Active lesions |                     |
|------------------------------|---------|-------------|------------------|--------|----------------|---------------------|
|                              |         |             | n <sup>1</sup>   | %      | n <sup>2</sup> | % of n <sup>1</sup> |
| 0-6 months                   | 3       | 3           | 3                | 100.00 | 3              | 100.00              |
| 6 months-3 years             | 17      | 17          | 17               | 100.00 | 12             | 70.59               |
| 4 years-12 years             | 13      | 13          | 13               | 100.00 | 11             | 84.62               |
| 13 years-18 years            | 4       | 4           | 4                | 100.00 | 1              | 25.00               |
| Females >18 years            | 62      | 61          | 37               | 60.66  | 6              | 16.22               |
| Males >18 years              | 54      | 51          | 43               | 84.31  | 4              | 9.30                |
| Sex ? >18 years <sup>3</sup> | 4       | 4           | 3                | 75.00  | 0              | 0.00                |
| Total                        | 157     | 153         | 120              | 78.43  | 37             | 30.83               |

<sup>1</sup> Refers to the number of individuals with CO.

<sup>2</sup> Refers to the number of individuals with "active" lesions of CO.

<sup>3</sup> Skeletons of indeterminant sex over the age of 18 years.

TABLE 3. Prevalence of cribra orbitalia and active lesions by age and sex from the Kellis cemetery

| Age cohort        | Total N | Assess-able | CO             |        | Active lesions |                     |
|-------------------|---------|-------------|----------------|--------|----------------|---------------------|
|                   |         |             | n <sup>1</sup> | %      | n <sup>2</sup> | % of n <sup>1</sup> |
| 0-6 months        | 54      | 33          | 7              | 21.21  | 6              | 85.71               |
| 6 months-3 years  | 53      | 31          | 22             | 70.97  | 15             | 68.18               |
| 4 years-12 years  | 28      | 20          | 18             | 90.00  | 7              | 38.89               |
| 13 years-18 years | 3       | 1           | 1              | 100.00 | 0              | 0.00                |
| Females >18 years | 50      | 35          | 21             | 60.00  | 3              | 14.29               |
| Males >18 years   | 36      | 23          | 9              | 39.13  | 1              | 11.11               |
| Sex ? >18 years   | 4       | 0           | —              | —      | —              | —                   |
| Total             | 228     | 143         | 78             | 54.55  | 32             | 41.03               |

<sup>1</sup> Refers to the number of individuals with CO.

<sup>2</sup> Refers to the number of individuals with "active" lesions of CO.

<sup>3</sup> Skeletons of indeterminant sex over the age of 18 years.

cally significant. Similarly, the K2 sample demonstrated no relationship among the sex of the individual, severity and occurrence of CO.

Thus, similarities and differences in CO characterize these two temporally distinct population samples. Individuals from ET show rates of 100% for the occurrence of CO in all childhood age cohorts. Although the prevalence of CO in K2 is also high, it is significantly lower than in ET. Upon reaching adulthood, females were similarly affected at both cemeteries whereas males were more highly affected at ET. Females in both samples show a high prevalence of active lesions, especially in ET.

## DISCUSSION

The human skeletal remains from the Dakhleh Oasis, Egypt are a unique series of superbly preserved skeletons showing exceptionally high rates of CO. Given the above trends, the interpretation of the rather obvious stress on these people is problematic for many reasons. In part this may be due to the geographic position of the Dakhleh Oasis. As the individuals from both cemeteries lived in northeastern Africa, this made them potentially susceptible to various environmental stressors that could favor the development of some sort of anemia. The possible factors include parasitic infestations (e.g., schistosomiasis), infectious diseases (e.g., one of the malaras), potentially low bioavail-



ability of iron in the diet, and even genetic anemias. Connected to the possibility of a parasite induced anemia is the model posed by Stuart-Macadam (1992) that such an anemic response by the host is actually an adaptive response. In short, a mild iron deficiency (hypoferremia) would be beneficial as this condition deprives the pathogen of the much needed iron that could be wrested from the host. Thus, according to this model, the presence of CO should actually be considered as an indicator of this adaptation.

Recently, this parasite model has been challenged by Holland and O'Brien (1997). Although they list many concerns in their review of the topic, they raise the point that hypoferremia may provide some benefit; however, the immune system would still be compromised by the chronic anemia. In other words, this "adaptation" to the presence of pathogens does so at the expense of a vast array of physiological reactions involved in maintaining homeostasis, such as collagen biosynthesis. It is important to consider that iron deficiency still exacts a physiological cost (for a review see Holland and O'Brien, 1997:185).

In this study, it is germane to discuss the prevalence of porotic hyperostosis (including CO) from areas adjacent to the study region. Unfortunately, there are no systematic surveys of the condition from other sites within the Oasis. Other areas of Egypt and Sudan have yielded studies of skeletal remains with variable rates of porotic hyperostosis (Batrawi, 1935; Satinoff, 1972a,b; Carlson et al., 1974; Strouhal and Jungwirth, 1980; Hummert and Van Gerven, 1983; Mittler and Van Gerven, 1994). None of these reports would serve as an appropriate comparison due to the geographic context of the Oasis. However, they do bring the Dakhleh remains into context for the region.

Mittler and Van Gerven (1994) updated a previous study of the early and late Christian cemeteries at Kulubnarti (Hummert and Van Gerven, 1983) by more closely examining the age specific trends of cribra orbitalia in remains spanning several time periods. The childhood specificity of CO is supported in their study, as in all studies. Additionally, they found that life expectancy

is reduced in individuals with the condition. Of significance in their study is that 100% of the individuals in the first year of life exhibit active lesions. This rate drops to 59% in children between 1 and 3 years, with a further decline in older children until the age of 12 years; beyond which all affected individuals exhibit healed lesions. They pose the question of whether this is evidence of an amelioration of the condition or whether the skeletal response has changed (Mittler and Van Gerven, 1994). An additional factor may be that the more intensive period of blood element formation has passed. Thus, the same conditions persist that caused the original anemia, however, the individual is not anemic to the point of affecting the marrow to the same extent as in childhood. They conclude that the pattern is consistent with the clinical pattern of iron deficiency anemia. In this case, the low bioavailability of iron in cereal grains (the largest portion of the diet), particularly those containing phytates, which act as iron chelators, and intensified parasitic infections causing gastrointestinal bleeding are all purported to result in the iron deficiency for this population.

The high prevalence of CO in young children characterizes both the Dakhleh and the Kulubnarti samples. However, the difference with the Dakhleh sample lies in the prevalence and severity for adults. It is possible then that there are some differences in the etiological factors underlying the condition in these populations. What evidence can be used to interpret the causes of CO in the Dakhleh population?

In an attempt to address the parasitic model, soil samples from the pelvic cavities of several individuals were examined for any signs of helminthic infestation. Although evidence of indigenous parasites were not found, this alone does not negate the possibility. Nonetheless, the possibility exists that the isolation of Dakhleh from the Nile Valley may be acting as a filter keeping parasitic infections to a minimum. Infant diarrheal diseases are still the primary health risk today in underdeveloped countries (Mensforth et al., 1978). Hrdlička (1912) notes that gastroenteritis was the major cause of death in the Kharga Oasis (adjacent

to the Dakhleh Oasis) in 1908. The etiology of infant gastrointestinal disorders is complex and multifactorial, involving the synergistic effects of malnutrition and infection. The infectious agents are often endogenous although, in some environments (e.g., endemic malaria) exogenous sources could play a significant role. Malaria must be considered a real factor in this region as it was documented in the Kharga Oasis by both Hoskins (1837, as cited by Hrdlička, 1912) and Hrdlička (1912). However, Hrdlička (1912:8) noted that "Malaria is not very frequent, except in the date season (September–October) when there are extraordinary numbers of flies and mosquitoes." Hoskins (1837 as cited by Hrdlička, 1912:8) further provided clues as to the general health of the Khargan inhabitants during his visit by describing them as "chiefly remarkable for the pallid and unhealthy hue of their countenances . . ." and having ". . . a languid and sickly appearance; a listlessness in their manner; a sluggishness in their movements; a total want of energy and vivacity." He further remarked on the "pallid hue" as being "most remarkable in their children and women." These descriptions indicate that the population of the Kharga Oasis was certainly of compromised health, particularly the children and women. But there is no way of knowing if their pallid constitutions reflect an underlying anemia.

As noted above, skeletal evidence of genetic anemias is absent on the Dakhleh skeletons (see Ortner and Putschar, 1985; Resnick and Niwayama, 1988; Hershkovitz et al., 1997; Aufderheide and Rodríguez-Martín, 1998). As a potential cause of the CO in the Dakhleh samples it is important to consider not only iron deficiency, but also folic acid and vitamin C deficiencies. When one of these nutrients is lacking, it directly affects the physiological status of the other two. For example, folic acid deficiency often accompanies a vitamin C deficiency. Vitamin C acts as an electron donor in the metabolism of folic acid (Stokes et al., 1975). A folic acid deficiency may lead to vitamin C and iron deficiencies. It is of interest to note that if goat's milk was being fed to infants, they would be getting a substantial proportion of ascorbic acid. It is the folic acid that would

be lacking in such a scenario (Forman, 1974 as cited by Janssens, 1983). Thus, evidence of compromised hemostasis in the form of a subperiosteal reaction in the orbits may be present without any of the other lesions indicative of scurvy. Two of the three nutrients, vitamin C and iron ( $\text{Fe}^{2+}$ ), are required in the hydroxylation of proline. The interrelatedness of these three nutrients dictates examination of the pathological changes to discern the likelihood of any or all three of these nutrients being deficient in the diet. In no instance have any of the intracranial remains from Dakhleh shown any evidence of the hemorrhaging, typically associated with vitamin C deficiency, regardless of age at death (Molto, 1986). Although Greenfield (1986) states that the pathological changes can be resorbed upon the amelioration of the deficiency, possibly yielding intracranial elements with no signs of ever having the condition, it is unlikely (given the intensity of the orbital lesions and their pervasive nature) that no individuals would show intracranial involvement.

In order to consider folic acid deficiency as an etiological agent for megaloblastic anemia, and thus the development of CO in infants, one must consider infant feeding practices. To that end one of the Dakhleh Oasis researchers (T. Dupras, McMaster University) is currently completing carbon and nitrogen stable isotope analysis of infant and juvenile remains from K2 in order to determine the timing of the weaning process and food(s) infants were weaned onto. Her analysis, with the addition of the dietary information, should further refine our understanding of the Dakhleh data relative to the folic acid hypothesis.

The cultural context (Roman-Byzantine) of these remains must be taken into consideration. Soranus (AD 98–117) favored maternal breastfeeding, however, he also believed that this should not begin before 3 weeks after birth of the infant in order to allow the mother time to regain her strength (Jackson, 1989). The recommended substitute for breastfeeding by the mother was a wet-nurse or even honey diluted with water or goat's milk. Both Galen and Soranus recommended a gradual weaning process; bread crumbled and softened with either

milk, hydromel (a mixture of honey and water), sweet wine or honey wine (all available during this time in the Dakhleh Oasis).

The introduction of both goat's milk and honey can have dire consequences for infants. Goat's milk is relatively low in both cobalamin (Cbl) and folate (0.1 µg/L and 6 µg/L, respectively) compared to human milk (4 µg/L and 52 µg/L, respectively) (Chanarin, 1990). Human infants that are started on goat's milk develop a severe megaloblastic anemia at about 3 to 5 months due to folic acid deficiency (Becroft and Holland, 1966). Normally the transfer of folate across the placenta is most active in the last weeks of pregnancy and fetal stores build-up is most evident after the 37th week (Chanarin, 1990). After birth there is normally a fall in red cell folate with the lowest levels being reached at 11–12 weeks. After 12 weeks, the fetal folate stores are exhausted and the newborn is dependent on dietary sources alone. A reduction in serum folate can result in iron malabsorption, hemopoietic marrow expansion and reduced levels of platelets and fibrinogen in the blood. The folate deficiency would have an effect almost immediately because of the low storage of folate in the infant. Chanarin (1990) notes that the WHO recommends that folate intake for the first 3 months of life should be 16 µg daily, between 3 and 6 months 24 µg daily and between 6 and 12 months 32 µg per day. The occurrence of active CO in these samples supports a case for the supplementation or dependence on goat's milk in infants less than 6 months of age. Such a situation would have likely resulted in a megaloblastic anemia.

As previously mentioned, honey has been implicated by the cultural context and documentary evidence from Soranus and Galen. Honey is a commodity that is listed within the Kellis Agricultural Accounts Book (Bag-nall, 1997). This document lists foods grown and traded in ancient Kellis and is roughly contemporary with the K2 remains in this study. Honey, when fed to infants, is a confirmed source of *Clostridium botulinum* spores. This saprophyte colonizes the infant's intestinal tract and forms a botulinal toxin resulting in botulism (Aron et al., 1979; Merenstein et al., 1991). Botulism is a

severe and often fatal form of food poisoning. A lethal dose for mammals is less than 1 µg/kg (Passmore and Eastwood, 1986). Botulinal toxin blocks transmission at neuromuscular junctions. Symptoms of onset within the first 6 months of life include apathy, weakness, constipation, floppiness, difficulty swallowing, sudden apnea (occasionally), ocular palsies, and, if unresolved, paralysis of the muscles of respiration leading directly to death (Passmore and Eastwood, 1986; Merenstein et al., 1991). With honey's confirmed presence in the Oasis at the time of the K2 sample and the possibility that weaning practices may have roughly followed those outlined by Galen and Soranus, it is reasonable to hypothesize that some, if not most, infants in the sample may have been compromised by botulism as well as megaloblastic anemia. Merenstein et al. (1991) note that most infants recover from the botulism after an illness that may last several weeks to months in modern populations. In the context of the inhabitants of ancient Kellis, recovery may not have been as well assured as in modern times.

In the case of an iron deficiency in adults, it is expected that young females will be more severely affected than males due to physiological and reproductive demands. There is no evidence for a difference in the prevalence or level of severity of the lesions between males and females at either cemetery. However, more females have active lesions than males in both samples, though it is more accentuated at ET. The pattern of an iron deficiency anemia being accompanied by a higher incidence of infection, as reported by Mensforth et al. (1978), is not well supported in this sample (see Cook et al., 1989; Molto, 1986, 2000). The apparent anemic reaction does not necessarily have to be exclusively attributed to an iron deficiency as other nutritionally-related deficiencies can also cause an expansion of marrow.

In contrast to the potential depletion rate of fetal folate stores, iron could take up to 4 or 5 months to deplete due to infant storage in the liver and the reticuloendothelial system of a full-term, healthy infant. Therefore, if infants less than 4 months were iron deficient they would not necessarily be ex-

pected to demonstrate any lesions. However, individual DK-31/39 (aged birth to 2 months) of the Dakhleh sample exhibits CO lesions in both orbits characterized by large and small foramina penetrating the bone cortex. It would seem, in this instance, a physiological demand for iron is not being met, producing an anemic reaction. As noted by Mensforth et al. (1978) clinical data indicate that iron deficiency is rare before 6 months of age, mainly because the newborn is afforded maternal protection. Yet, in the ET sample 100% (3/3) of the infants in the birth to 6 month age cohort exhibit active CO; whereas the rate for K2 is 21% (7/33). This indicates that, at the very least, these children were not born with suitable stores to protect them from the anemic stress.

### CONCLUSIONS

The ET cemetery provides evidence of a population that was more severely compromised than the later K2 group. The ethnographic material concerning the inhabitants of the adjacent Kharga Oasis by Hoskins (1837) and Hrdlicka (1912) provide tantalizing support for the argument that the anemic condition of these people was contributed to by gastroenteritis possibly exacerbated by malaria. However, the awaited stable isotope data may resolve the question as to whether or not the infants were being weaned before the age of 6 months on goat's or cow's milk. Given the early onset of lesions, it is more likely that it was goat's milk that was being used, and this led directly to the development of megaloblastic anemia in the infants. This notwithstanding, malaria and even gastroenteritis cannot be eliminated as possible sources of anemic stress. Further, the introduction of honey as a dietary supplement possibly resulted in cases of botulism in infants.

Cultural differences in the diet cannot be ruled out as a possible explanation for the difference seen in the infant remains from both cemeteries. Such cultural differences could include differences in weaning practices and a change in dietary components or proportions of dietary constituents. Other factors such as malaria and gastroenteritis may have also changed in their prevalence

in the Oasis over time. For example, today it is reported that there has not been a case of malaria since the 1940's (A.J. Mills, personal communication, 1990).

The inhabitants of ancient Dakhleh present a remarkable opportunity to study their dietary habits in light of direct documentation of foods grown in the Oasis, as well as exceptional preservational quality of plant, animal and human remains for palaeopathological and palaeonutritional analyses.

### ACKNOWLEDGMENTS

The authors express their appreciation to Mr. Tony Mills, Director of the Dakhleh Oasis Project, Dr. Peter Sheldrick, Ms. Megan Cook, and Mr. Alan Hollett for all their assistance in the field. The authors also thank Ms. Tracy S. Oost for producing the map in this publication. Dr. Fairgrieve was supported by a two-year postgraduate doctoral scholarship from the Natural Sciences and Engineering Research Council of Canada (NSERC), a University of Toronto Open Doctoral Fellowship, and further travel funds by the Laurentian University Research Fund. This research was also facilitated in part a grant held with Dr. Henry Schwarcz from the Social Sciences and Humanities Research Council (No. 410-94-0857). Dr. Molto's research in the Oasis has been supported by several small grants from the Lakehead University Senate Research Committee. Helpful editorial comments were made by Dr. Henry Schwarcz and Ms. Tosha Dupras, to whom we are grateful.

### LITERATURE CITED

- Angel JL. 1964. Osteoporosis: Thalassemia? *Am J Phys Anthropol* 22:369-374.
- Angel JL. 1971. *The People of Lerna*. Washington, D.C.: Smithsonian Institution Press.
- Arnon SS, Midura TF, Damus K, Thompson B, Wood RM, Chin J. 1979. Honey and other environmental risk factors for infant botulism. *J Pediatrics* 94:331-336.
- Aufderheide AC, Rodríguez-Martín C. 1998. *The Cambridge encyclopedia of human paleopathology*. Cambridge: Cambridge University Press.
- Bagnall RS. 1997. *The Kellis agricultural account book (P. Kell. IV. Gr. 96) Dakhleh Oasis Project Monograph 7*. Oxford: Oxbow.
- Bass WM. 1987. *Human osteology: A laboratory and field manual*. 3rd ed. Columbia MO.: Missouri Archaeological Society.
- Batravi AM. 1935. *Report on the human remains, mission Archéologie de Nubie 1929-1934*. Cairo: Service des Antiquités du L'Egypte, Government Press.



- Brocroft DMO, Holland JT. 1966. Goat's milk and megaloblastic anaemia of infancy: a report of three cases and a survey of the folic acid activity of some New Zealand milks. *New Zealand Med J* 65:303-307.
- Brooks S, Suchey JM. 1990. Skeletal age determination based on the os pubis: a comparison of the Acsádi-Nemeskéri and Suchey-Brooks methods. *Hum Evol* 5:227-238.
- Butzer KW. 1976. *Early hydraulic civilization in Egypt*. Chicago: University of Chicago Press.
- Carlson DS, Armelagos GJ, van Gerven DP. 1974. Factors influencing the etiology of cribra orbitalia in prehistoric Nubia. *J Hum Evol* 3:405-410.
- Chanarin I. 1990. *The megaloblastic anemias*, 3rd ed. Oxford: Blackwell Scientific Publications.
- Churcher CS. 1983. Dakhleh Oasis Project palaeontology: Interim report on the 1982 field season. *J Soc Stud Egypt Antiq* 13:178-187.
- Cohen MN, Armelagos GJ, editors. 1984. *Paleopathology at the origins of food production*. Toronto: Academic Press.
- Cook M, Molto E, Anderson C. 1988. Possible case of hyperparathyroidism in a Roman period skeleton from the Dakhleh oasis, Egypt, diagnosed using bone histomorphometry. *Am J Phys Anthropol* 75:23-30.
- El-Najjar MY, Ryan DJ, Turner CG, Lozoff B. 1976. The etiology of porotic hyperostosis among the prehistoric and historic Anasazi Indians of southwestern United States. *Am J Phys Anthropol* 20:329-337.
- El-Najjar MY. 1976. Maize, malaria, and the anemias in the pre-Columbian New World. *Ybk Phys Anthropol* 9:75-78.
- Fairgrieve SI. 1993. Amino acid residue analysis of type I collagen in human hard tissue: an assessment of cribra orbitalia in an ancient skeletal sample from tomb 31, site 31/435-D5-2, Dakhleh Oasis, Egypt. PhD Thesis, Department of Anthropology, University of Toronto.
- Greenfield GB. 1986. *Radiology of bone diseases*, 4th ed. Philadelphia: JB Lippincott.
- Guidotti A. 1984. Frequencies of cribra orbitalia in central Italy (19th century) under special consideration of their degrees of expression. *Anthropol Anz* 42:11-16.
- Henneberg M, Steyn M. 1994. Preliminary report on the paleodemography of K2 and Mapungubwe populations (South Africa). *Hum Biol* 66:105-120.
- Hershkovitz J, Rothschild BM, Latimer B, Dutour O, Léonetti G, Greenwald CM, Rothschild C, Jellema LM. 1997. Recognition of sickle cell anemia in skeletal remains of children. *Am J Phys Anthropol* 104:213-226.
- Holland TD, O'Brien MJ. 1997. Parasites, porotic hyperostosis, and the implications of changing perspectives. *Amer Antiq* 62:183-193.
- Hoskins GA. 1837. *Visit to the Great Oasis of the Libyan Desert*. 8°, London.
- Hrdlička A. 1912. The Natives of Kharga Oasis, Egypt. *Smithsonian Misc Collect* 59:1-153.
- Hummert JR, Van Gerven DP. 1983. Skeletal growth in a medieval population from Sudanese Nubia. *Am J Phys Anthropol* 60:471-478.
- Jackson R. 1989. *Doctors and diseases in the Roman Empire*. Norman, OK: University of Oklahoma Press.
- Janssens PA. 1983. Porotic hyperostosis and goat's milk anaemia: A theory (more). *OSSA* 8:101-108.
- Katz DM, Suchey JM. 1986. Age determination of the male os pubis. *Am J Phys Anthropol* 69:427-435.
- Mensforth R, Lovejoy CO, Lallo J, Armelagos GJ. 1978. The role of constitutional factors, diet and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Med Anthropol* 2:1-59.
- Merenstein GB, Kaplan DW, Rosenberg AA. 1991. Silver, Kempe, Bruyn and Fulginiti's handbook of pediatrics. Norwalk, CT: Appleton and Lange.
- Mills AJ. 1979. Dakhleh Oasis Project: report on the first season of survey, October-December, 1978. *J Soc Stud Egypt Antiq* 9:163-185.
- Mittler DM, Van Gerven DP. 1994. Developmental, diachronic, and demographic analysis of cribra orbitalia in the Medieval Christian population of Kulubnarti. *Am J Phys Anthropol* 93:287-297.
- Molto JE. 1986. Dakhleh Oasis Project: Human skeletal remains from the Dakhleh Oasis, Egypt. *J Soc Stud Egypt Antiq* 16:119-127.
- Molto JE. 2000. The comparative skeletal biology and palaeoepidemiology of the people from 'Ein Tirghi and Kellis, Dakhleh Oasis, Egypt. In: Mills T, Marlow M, editors. Oxford: Oxbow Press.
- Moseley JE. 1974. Skeletal changes in the anemias. *Sem Roentgenol* 9:169-184.
- Nathan H, Haas N. 1966. On the presence of cribra orbitalia in apes and monkeys. *Am J Phys Anthropol* 24:351-360.
- Ortner DJ, Putschar WGJ. 1985. Identification of pathological conditions in human skeletal remains, *Smithsonian Contributions to Anthropology* 28. Washington, DC.: Smithsonian Institution.
- Palkovich AM. 1987. Endemic disease patterns in paleopathology: Porotic hyperostosis. *Am J Phys Anthropol* 74:527-537.
- Passmore R, Eastwood MA. 1986. *Davidson and Passmore human nutrition and dietetics*. Edinburgh: Churchill Livingstone.
- Phenice TW. 1969. A newly developed visual method of sexing the os pubis. *Am J Phys Anthropol* 30:297-302.
- Resnick D, Niwayama G. 1988. *Diagnosis of bone and joint disorders*. Philadelphia: W.B. Saunders.
- Satinoff MI. 1972a. The origins and geographical spread of the thalassemias and abnormal haemoglobins. *J Hum Evol* 1:79-82.
- Satinoff MI. 1972b. The medical biology of the early Egyptian populations from Asswan, Assyut, and Gebelen. *J Hum Evol* 1:247-257.
- Schwarcz HP, Dupras TL, Fairgrieve SI. 1999.  $^{15}\text{N}$  enrichment in the Sahara: in search of a global relationship. *J Archaeol Sci* 26:629-636.
- Steinbock RT. 1976. *Paleopathological diagnosis and interpretations*. Springfield, IL: Charles C. Thomas.
- Stokes PL, Melikian V, Leeming RL, Portman GH, Blair JA, Cooke WT. 1975. Folate metabolism in scurvy. *Am J Clin Nutr* 28:126-129.
- Strouhal E, Jungwirth J. 1980. Paleopathology of the Late Roman-Early Byzantine cemeteries at Sayala, Egyptian Nubia. *J Hum Evol* 9:61-70.
- Stuart-Macadam P. 1982. A correlative study of a palaeopathology of the skull. PhD thesis, Department of Physical Anthropology, University of Cambridge.
- Stuart-Macadam P. 1985. Porotic hyperostosis: representative of a childhood condition. *Am J Phys Anthropol* 66:391-398.
- Stuart-Macadam P. 1987. Porotic hyperostosis: New evidence to support the anemia theory. *Am J Phys Anthropol* 74:521-526.
- Stuart-Macadam P. 1989. Porotic hyperostosis: Relationship between orbital and vault lesions. *Am J Phys Anthropol* 80:187-193.
- Stuart-Macadam P. 1992. Porotic hyperostosis: A new perspective. *Am J Phys Anthropol* 87:39-47.
- Ubelaker DH. 1978. *Human skeletal remains: Excavation, analysis, interpretation*. Washington, D.C.: Taraxacum.
- Walker PL. 1986. Porotic hyperostosis in a marine-dependent California Indian population. *Am J Phys Anthropol* 69:345-354.